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"Discuss the rationale and scientific basis for the continued use of water fluoridation in communities where caries risk has declined due to the 'halo effect' of other fluoride sources."

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Undergraduate competition winning essay 2005

Abstract

Community water fluoridation is widely recognised as the most effective and successful public health measure implemented against tooth decay. However, there has been an emergence of evidence over the past few decades demonstrating dilution and diffusion phenomena or 'halo effect' as well as data supporting the so-called 'secular decline' of caries diminishing the apparent effectiveness of water fluoridation. In light of this, it is necessary to re-evaluate available scientific literature and subsequently develop a cogent and scientifically sound rationale to justify sustaining the fluoridation of communal water supplies.

Introduction

Artificial fluoridation of communal water supplies has been implemented for over half a century. Since the pioneer fluoridation program in Grand Rapids, Michigan, there has been an increasing assemblage of epidemiological evidence demonstrating and supporting the benefits imparted by water fluoridation. While community Water fluoridation has met with opposition from its inception, it remains an imperative component of public heath efforts aimed at prevention of dental disease. This literature review will address the rationale and scientific evidence for its continued effectiveness and use in caries prevention and success in the realm of public health.

The effectiveness of community water fluoridation in caries prevention

It is pertinent to firstly discuss the issue of effectiveness of water fluoridation, and its role as a major contributor to the scientific reasoning for the continuation of such programs. The effectiveness of fluoridation of community water supplies is well established, with numerous studies undertaken worldwide throughout the decades, supporting its efficacy in reducing dental caries. Murray and Rugg-Gunn reviewed 95 studies between 1945 and 1978 and reported caries reductions following implementation of community water fluoridation in the order of 40-50% in the deciduous and 50-60% in the adult dentitions.[1] In 1989 Newbrun conducted a review of the efficacy of water fluoridation based on surveys of caries prevalence in non-fluoridated and fluoridated communities during the period 1979-1989 in the United States, Australia, Britain, Canada, Ireland and New Zealand. Results demonstrated a reduction of caries prevalence between fluoridated and non fluoridated communities in the range of 30-60% for deciduous dentition and 15-35% in the adult dentition. Newbrun concluded that water fluoridation demonstrated a *consistent benefit* to both deciduous and permanent dentitions, resulting in substantially lower caries prevalence in fluoridated areas.[2]

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THIS ISSUE

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1996 marked the 50th year after the first public water fluoridation project was announced, and an International Symposium was held where several countries around the world submitted reports of their experience with community water fluoridation. The United States concluded after 50 years of experience with water fluoridation that it 'dramatically reduces the incidence and prevalence of dental caries'.[3] Studies in Ireland recognised that 'children and adolescents experience less dental caries and adults retain more of their natural teeth if they reside in communities served with fluoridated water supplies'.[4] The success and effectiveness of water fluoridation was also documented in Australia, Singapore and South Africa. [5,6,7]

Current studies conducted in Australia have established that a *continued* effectiveness of fluoridation in caries prevention exists. Recent research in Western Australia comparing the water-fluoridated Perth with unfluoridated Bunbury found a statically significant difference between caries experience levels,[8] supporting results of a similar study conducted by Slade *et al* and one several years prior to it by Stockwell *et al*.[9,10]

These in addition to numerous other studies[11,12] provide consistent and sound evidence for the efficacy of water fluoridation in caries prevention since its implementation in 1945 to the present, adding weight to its scientific basis and rationale for its continuation.

The 'dilution' and 'diffusion' phenomena in relation to community Water fluoridation

It has become apparent over the past three decades that the range of caries reduction when comparing fluoridated to non-fluoridated communities has decreased in magnitude, in comparison to when water fluoridation was first introduced. While the difference is still significant, figures have shown 20-40% less decay in fluoridated communities from 1979-1989 in comparison to a 40-60% difference between 1945-1978.[1,2,10] Ripa's review on water fluoridation attributed the apparent diminishing effectiveness of water fluoridation to two main factors, known as dilution and diffusion, or the so-called 'halo effect'.[13]

When water fluoridation programs were first introduced for caries prevention purposes, it was the only significant source of fluoride exposure.[14,15] However, other forms of fluoride have become widely available since then in the form of fluoride tablets, mouthrinses, dentifrices and gels among others, resulting in a fall in caries prevalence in communities lacking water fluoridation. This is especially the case in industrialised countries. This decrease has been attributed mostly to the availability use of fluoride-containing dentifrices, particularly since the1970s.[16,17] The widespread use of these other fluoride vehicles, used in both fluoridated and non-fluoridated areas, has resulted in a diminished difference in the caries prevalence between fluoridated and non-fluoridated communities. This is referred to as the '*dilution effect*' on the apparent effectiveness of water fluoridation.[5, 17,19]

The phenomenon of 'diffusion' occurs when foods and beverages processed in fluoridated areas (and therefore containing fluoride) are distributed for consumption in non-fluoridated communities. Persons who consume these products will then ingest the fluoride resulting in a disseminated or *diffusion effect* of fluoridated water in the prevention of dental decay[17,19] resulting in an attenuation of the apparent effectiveness of water fluoridation.

It is reasonable then, to consider the effectiveness of water fluoridation in light of the 'dilution' and 'diffusion' effects, and discuss the rational behind continuing such programs despite these phenomena. It is essential firstly to assert that although the efficacy of water fluoridation appears to have diminished as a result of diffusion and dilution, the approximately 20% difference that still remains between fluoridated and non-fluoridated communities is still a substantial figure[17], which on its own is a significant factor in the justification for the continuation of such programs. In addition to this, many recent and current studies support the continued benefit of water fluoridation. Secondly, diffusion and dilution do not influence the intrinsic effectiveness of water fluoridation, that is to say, its caries-preventive effects are still the same as at the time of its inception if introduced to a population not exposed to other sources of fluoride by dilution or diffusion.[18]

The diffusion effect is a direct outcome of water fluoridation that has, almost ironically, resulted in an apparent decrease in the effectiveness of water fluoridation. Rather, the diffusion effect should be viewed as a beneficial sequelae to water fluoridation, where persons living in non-fluoridated communities may derive the benefits of water fluoridation, rather than being a contraindication to the continuation of fluoridation programs. Griffin *et al* concluded that a failure to account from the diffusion effect of water fluoridation results in an underestimation of the total benefit of water fluoridation. Their results found that US children living in non-fluoridated areas with low diffusion exposure experienced higher levels of dental caries than children who lived in

Table 1. Percentage difference of caries prevalence according to community fluoridation status.

Author, year of survey	Age (years)	% difference in DMFS/DMFT between optimally and low fluoridated communities	Location
O'Mullane *et al* 1988	5 8 12 15	40 40 21 24	Ireland
Rugg-Gunn *et al*	5 5	54 54	Northumberland Newcastle
Brunelle and Carlos 1990	5 8 15	39 8 18	United States
Stockwell *et al* 1990	15	41	Australia
Riordan 1991	12	43	Australia

non-fluoridated areas with a high exposure to diffusion effects.[21] Clearly, the phenomenon of diffusion would cease to exist if water fluoridation was discontinued, as well as the benefits associated with it.

The 'dilution' of the benefits of water fluoridation by other vehicles of fluoride delivery cannot be contested; however, it has also highlighted the need to determine whether the use of other fluoride vehicles obviates the need for water fluoridation, as well as the relative benefits in caries prevention of these various fluoride sources in comparison to water fluoridation.

A cross sectional study that took place in Western Australia attributed the difference in caries prevalence between the Busselton-Bunbury area and Perth were most likely due to the differences in the levels of fluoride in the water supplies, rather than differences in, for example, fluoride-containing dentifrices.[10] In the same year, another study found that US children who had never lived in a fluoridated community had mean DMFS scores 18% higher than those who had lived in a fluoridated community. Upon controlling for dilution effects, this difference increase to 25%.[22] Both studies suggest that water fluoridation exerts caries preventive effects *in addition* to those provided by other fluoride sources – thus, water fluoridation is not made redundant by the use of other fluoride sources. Furthermore, there is some evidence that when water fluoridation and fluoride dentifrices are used together, the caries-preventive effects are additive.[2] Hence, rather than one obviating the use of the other, it is most likely that the benefit attained from utilising both fluoridated water and dentifrices would be greater than exposure to only one of these.

A study by Spencer addressing the dilution effect quantified the association of fluoridated water supplies, fluoride supplements and fluoride containing dentifrice with caries severity in Australian adolescents between 1965-1978. The results indicated that decline in caries severity was most associated with fluoridated water.[10]

Hence, despite the so-called dilution effect, the continuing effectiveness of fluoridated water and it's benefits in addition to other fluoride sources are a sound basis for it's continuation.

Continuing fluoridation of community water supplies: a public health perspective

The World Health Organisation has endorsed community water fluoridation as the most effective public health measure for the prevention of dental decay.[23] Community water fluoridation possesses many attributes that make it an ideal vehicle for a public health approach aimed at dental health, in terms of economics, efficacy, equity, safety and compliance.[24]

From an economic standpoint, water fluoridation is a relatively inexpensive exercise, with the annual average cost per person approximately US $0.50 – the accumulated costs over a lifetime would only equal the cost of a single restoration or less.[3] The cost-effectiveness of community water fluoridation programs has been well documented worldwide in countries such as Singapore, Australia, the United States and Ireland.[3,4,5,6]

As previously addressed, the efficacy of water fluoridation has been studied extensively via epidemiological research worldwide and is widely acknowledged as having consistent, reliable benefits making it an effective public health measure. A recent study demonstrated a continued effectiveness of water fluoridation and also found that community water fluoridation was likely to exert a greater public health impact than replacing it with dental sealant placements, programs providing dietary fluoride supplements and professionally applied fluoride treatments.[27]

Water fluoridation has the distinct advantage of being an equitable means of disease prevention. The caries-preventive effect of fluoridated water supplies benefit all segments of the population, regardless of age, social-economic status or other social variables.[18] Fluoride exerts both pre- and post-eruptive mechanisms of caries prevention. Pre-eruptively, fluoride ingested systemically is incorporated into the developing enamel producing fluoroapatite crystals, which are more resistant to decay than their hydroxyapatite counterpart. The post-eruptive mechanism is largely attributed to it's topical effect on enamel, enhancing remineralization and inhibiting demineralisation. The amount of fluoride added to water is sufficient for such a therapeutic effect.[25] As a result, the benefit of water fluoridation may be derived at all ages with it continuing for a lifetime. A abundance of research is available demonstrating the efficacy of water fluoridation on decreasing caries prevalence in children, and there is increasing evidence suggesting a preventive effect on root caries in the adult population.[18]

Research has also found that community water fluoridation is effective regardless of socio-economic status (SES), and is able to overcome or at least reduce socio-economic inequalities with regards to burden of disease. While there are fluoride vehicles other than of water supplies available that 'dilute' the effectiveness of water fluoridation, there are variations in the exposure to these sources. A review by Horowitz stating that 'many poor children do not have fluoride toothpastes in their homes, do not receive professional preventive services, and are not likely to take dietary fluoride supplements'[18] demonstrates the presence socio-economic inequalities and the resultant effect on access to other fluoride vehicles very clearly. Various studies in Australia and the United Kingdom came to the conclusion that children of lower SES generally had more caries experience, and that SES inequalities remained despite all children receiving free dental services. However, SES inequalities were reduced among children with exposure to water fluoridation.[5,27] Hence water fluoridation represents a socially equitable public health measure due to its ability to exert caries preventive effects in all socio-economic groups and also reduce existing socio-economic inequalities in caries experience.[27]

Another considerable advantage of using water fluoridation as a public health measure lies in the elimination of the necessity for compliance.[18,25] Water fluoridation is effective regardless of an individual's motivation for maintaining optimal home care and desire to use and pay for dental services. The fact that water is an essential dietary component of all members of the community[23] is an inherent attribute that also contributes to the success of using community water supplies as a fluoride vehicle.

In terms of safety, 'no other disease preventive method has been studied as extensively for safety'. Research on disease incidence and prevalence, morbidity and mortality on water

fluoridation have affirmed the safety of fluoridation of community water supplies.[18, 28] Fluorosis is an issue that is frequently referred to by those opposing fluoridation. However, findings from a recent study demonstrated that there was a narrowed prevalence of dental fluorosis between non-fluoridated and fluoridated communities[27] with dental fluorosis increasing proportionally more in non-fluoridated than fluoridated communities over the past 5-6 decades.[26] Other studies have shown that the principal contributors to the increased prevalence to fluorosis are the early use of fluoride toothpastes, the use and misuse of dietary fluoride supplements and the prolonged consumption of infant formula.[18, 29] It has been estimated that the portion of fluorosis due to water fluoridation is 40%, less than that attributed to other fluoride sources (60%).[20]

When considering water fluoridation from a public health standpoint, it is pertinent to discuss the so-called 'secular decrease' in caries experience. It is widely accepted that in the past 25 years the prevalence of dental caries has declined in most industrialised countries raising a valid question of whether water fluoridation is still necessary as a public health measure, especially when the condition being prevented is less prevalent.[17,19] There is no one unequivocal explanation for this decrease although it has largely been attributed the widespread use of fluoride dentifrices.[17,30] To answer this question it is important to take into account the following; firstly, the fact remains that dental caries is the single most common disease of childhood that is not self-limiting or manageable by antibiotics.[24] In developing countries, caries prevalence is on the increase, and furthermore, this 'secular decline' has not continued during the 1990s, and it is possible that it has reached a plateau.[31] The World Health Organisation in 2001 described caries as a 'worldwide epidemic'[23] and recently, the World Oral Health Report made in 2003 stated that dental caries remains a major public health problem even in industrialised countries, affecting 60-90% of school children as well as a great majority of adults.[32] Taking these facts into consideration it is very apparent that water fluoridation is still a necessary public health measure.

The repercussions after cessation of community water fluoridation

An alternative and useful way of approaching the question whether water fluoridation programs should be continued, is to observe the sequelae of its cessation. In Karl-Marx-Stadt (now Chemnitz, Germany) there had been a significant caries reduction subsequent to the implementation of water fluoridation in 1959. However, in the following years the city experienced irregularities in the fluoride concentration of the drinking water, and consequently had corresponding fluctuations of caries experience – increased and decreased concentrations of fluoride resulted in markedly decreased and increased DMFT indices respectively. After fluoride levels were returned to optimal concentrations, the caries prevalence was returned to what had been achieved previously. It was concluded that caries prevalence was directly linked to the level of water fluoridation.[33]

Another study conducted in Scotland demonstrated that five years after cessation of water fluoridation of Stranraer, the caries prevalence had increased to nearly equivalent that of the non-fluoridated Annan. The authors concluded that water fluoridation still provided the benefit of caries prevention despite the 'general decline in dental caries'.[34]

While most studies of this nature, including the two above, have demonstrated that long-lasting discontinuities or complete cessation of community water fluoridation have resulted in a caries increase, the results of some recent literature appear to demonstrate the reverse. However, interpretation of these results must be made with caution.

The discontinuation of water fluoridation in La Salud, Cuba seemed to have no effect on the city's caries index, with DMFT remaining stable or even decreasing. However, it should be noted that the study population had also been provided with a school mouthrinsing program with a 0.2% NaF solution.[35]

In the city of Kuopio, Finland, discontinuation of water fluoridation resulted in no increase of caries frequency in primary teeth. However, the children in this community were also concurrently receiving comprehensive dental care.[36]

A cross sectional study taking place in former East Germany found that subsequent to cessation of water fluoridation there was a decrease in caries index. However, the German national data revealed that a noteworthy 90.7% of the population visited the dentist at least once a year, 40.3% of subjects had fissure sealants placed in their mouths, fluoride-containing dentifrices were used by 88% of the population, fluoridated salt was available and children received vitamin D tablets combined with fluoride in the first year of life.[37]

It is therefore obvious that these studies do not provide convincing results to demonstrate a redundancy of water fluoridation in terms of caries prevention, due to the variables that exist. The cessation of water fluoridation is, at this stage, still associated with a detrimental effect on a community's caries experience, when no other confounding factors apply.

Conclusion

There is a plethora of literature available spanning greater than 50 years addressing community water fluoridation and its effect on dental caries, as well as from a public health perspective. The collective results obtained from the available research provide consistent evidence as well as compelling public health arguments for the implementation of community water fluoridation, whilst providing convincing scientific basis and rationale to justify its continuation worldwide.

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In-office fluoride products: current concepts for maximal effectiveness (part 1)

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This article is the first in a two part series which discuss current concepts in the use of fluoride products. The second part will address products for at-home use.

In office fluoride therapies comprise products designed for topical application (such as fluoride solutions, gels, foams and varnishes), materials which release fluoride and can be recharged (based on glass ionomer cements), fluoride combined with other therapeutically active materials (such as silver or Recaldent), and techniques for enhancing fluoride uptake into tooth structure (such as iontophoresis and laser activation).

In-office fluoride gel therapy is practised widely by dentists and dental auxiliaries, and has been regarded as a mainstay of general practice for many years, contributing a reduction in the order of 25% or greater in caries increment. This benefit has been derived from the formation of various fluoride compounds on the tooth surface, leading to the presence of small quantities of fluoride in saliva and plaque fluid over extended periods, a concept which is termed 'loading the intra-oral fluoride reservoir'. Best practice in the use of professionally-applied fluoride therapies requires simple and logical protocols which maximise benefits for patients while reducing risks of toxicity or other complications.

Fluoride gels and foams

Historically, the mainstay of professional fluoride application in Australia for many years has been the annual or semi-annual application of a concentrated fluoride product (9,000-12,300ppm F, equivalent to 9-12.3 mg/g) such as neutral sodium fluoride (NaF) or acidulated phosphate fluoride (APF) immediately following a prophylaxis. Some caveats relating to this practice are outlined below.

Dental plaque has been shown to uptake fluoride readily and to serve as a fluoride reservoir. Only very thick plaque deposits (i.e. visible to the naked eye) appear capable of impairing the eventual uptake of fluoride from a gel into the enamel surface. Unless visible plaque is present, there is therefore no absolute requirement for a professional prophylaxis prior to gel application. If a prophylaxis is judged to be necessary, a paste with low abrasivity should be used to avoid removing the pellicle layer or abrading the enamel surface. Interestingly, most dentifrices do not appear capable of removing pellicle and would therefore be suitable for use as an alternative to prophylaxis pastes.

When high concentration gels are applied to the tooth surface, calcium fluoride (CaF_2) and other materials are precipitated in a layer of some 20 microns in thickness. The CaF_2 dissolves slowly over the following days, contributing fluoride to the intra-oral reservoir. This loading effect is the reason that vigorous rinsing after topical fluoride applications in the dental surgery is not advised. The precipitation process occurs in an exponential fashion, with much of the deposition occurring in the first minute after application, although even thicker layers are formed after 4 minutes.

In the moderate caries risk patient, a suitable protocol for prophylactic application of fluoride gel or foam would be to use 1-2mL applied at 6 or 12 month intervals for a period of 4 minutes. For a high risk patient, fluoride varnish applied at 3-4 month intervals would be used instead, in combination with a tailored home fluoride program, as will be discussed in the second article.

The author prefers neutral NaF gel to APF gel for a single in-office application because the material is better tolerated by patients and there is less risk of hypersalivation which could lead to accidental ingestion. If patients have just undergone periodontal debridement, the use of a neutral NaF product will not affect the smear layer formed by the scalers, curettes and other instruments used, whilst APF can erode away all or part of this smear layer and thus contribute to post-debridement dentinal hyper-sensitivity. The low pH (~2-3) and high titratable acidity of APF can contribute to erosive damage of glass ionomer restorations and the glass-based fillers in composite resin, as well as the loss of glaze of porcelain restorations, if used excessively (such as may occur if used daily at home). There published evidence on efficacy of fluoride gels shows that similar levels of caries prevention (in DMFS) occur with APF, neutral NaF, and stannous fluoride. Because the low pH of APF products gives a strong acidic taste which provokes marked salivation, significant ingestion of fluoride may result from inadvertent swallowing by children. Ingested F reacts with gastric HCl to give HF (hydrofluoric acid), which causes irritation to the gastric mucosa. (This explains why milk is an antidote for excessive ingestion of F, since under acidic conditions, casein phospho-peptides in milk will release calcium ions which will bind F ions into CaF_2). Ingestion is a particular issue if fluoride gel is used in moderate amounts and in an uncontrolled bulk delivery system (such as a stock single or double-arch tray, or when applied with a toothbrush).

For patient comfort and safety, and particularly to prevent hypersalivation and possible nausea, neutral NaF gel is preferred when a risk assessment determines that the patient is at an enhanced risk of developing dental caries. If trays are considered desirable, the most useful designs are foam-lined, hinged trays that allow both arches to be treated at the same time. Trays should cover all surfaces of the teeth, be comfortable and have high extensions into the mucobuccal fold. Patients should be seated up-right during application, and have low velocity suction in place during the 4 minute treatment time, with additional saliva control using adhesive absorbent pads on the parotid ducts if convenient. Immediately after the trays are removed, high velocity suction should be applied to remove excess gel, and the patient encouraged to

expectorate any saliva and excess gel (but not rinse). The patient should not eat or drink for 30 minutes afterwards.

Because of the concern with ingestion, fluoride gels and foams should only be used in older children (aged 10 and above) and in adults. For children less than 10 years of age with a high caries risk, fluoride varnishes should be used rather than fluoride gels, as the benefit/risk ratio is much higher because of the slow release properties of varnish and the small volumes used. This is illustrated in the following calculations. For a 10kg child (12 months old), the Probable Toxic Dose of 5mg F/kg body weight would require the rapid ingestion of 5mL of neutral NaF gel or 1.1mL varnish. The latter cannot occur as varnish is only applied in volumes of 0.2mL and the fluoride is released over several days rather than rapidly. By comparison, in a 70kg adult, the Probable Toxic Dose of 350 mg F would require the rapid ingestion 40 mL of neutral NaF gel (i.e. more than one 30mL bottle) or 8.8mL of varnish.

As a final point, fluoride gels and foams should not be mixed with other preventive products (such as chlorhexidine gels or pastes based on Recaldent) since this may lower the efficacy of both ingredients in the mix because of chemical interactions such as the formation of insoluble compounds which then precipitate.

Fluoride varnishes

These are an excellent means for achieving a medium term delivery of fluoride onto sites of enamel or root surfaces affected by incipient caries. Clinical trials show reductions in caries incidence ranging from 18 to 70%. Fluoride varnishes contain the highest levels of fluoride of any therapeutic product (22,600-25,000 ppm). A concentration of 5% NaF by weight gives 22,600 ppm of fluoride, which is the level found in Duraphat™, the most widely used fluoride varnish. Fluor-Protector™ contains 0.9% by weight fluor-silane (7,000 ppm F).

Because of these high concentrations, fluoride varnishes are able to exert powerful anti-bacterial actions by:

- impairing glycolysis, energy utilisation and other metabolic processes within dental plaque bacteria;
- through formation of the compound HF which acidifies the interior of bacterial cells;
- inactivating metabolic enzymes;
- interfering with bacterial membrane permeability to ionic transfer;
- inhibiting the synthesis of intracellular polymers; and
- by inhibiting key enzyme systems such as enolase and H+/ATPase.

Importantly, high concentrations of F (0.16-0.3mol/L, equivalent to 3,000-5,600 ppm) will kill dental plaque, and thus provide a chemical plaque control effect, albeit for a limited time period immediately after application.

The clinical application protocol for fluoride varnish application should account for the fact that maximal benefit occurs with repeated application several times per year in the high risk patient. A frequency of every 3 months aligns with a suitable recall interval for high risk patients, such as those with profound salivary dysfunction. As noted above for fluoride gels and foams, professional prophylaxis before varnish application is not necessary. The slow release of fluoride from varnish not only gives a sustained benefit over many days but also gives an excellent safety profile since the amount released (and thus available for ingestion) is very low. Their high effectiveness, ease of use, and benefit/safety ratio give fluoride varnishes a major advantage over in-office fluoride gel and foam treatments. Fluoride varnishes are the recommended means for applying topical fluoride in children aged less than 10 years, as gels and foams are contra-indicated. A typical full-mouth application would require a maximum of 0.2mL of varnish, which would be applied with cotton pellet, cotton pellet, sponge-tipped applicator or brush.

Fluoride varnish is a highly cost-effective treatment for selected 'at risk' surfaces when evidence suggests that the patient is highly caries susceptible (e.g. the patient has a high number of DMFS for their age, high levels of fermenting bacteria in their plaque, poor dietary and lifestyle factors, and no history of systemic fluoride exposure). There is, even in the absence of clinically active disease, some justification for concentrated for 'at risk' surfaces. For example, a 12-year-old child, with multiple posterior proximal restorations has a number of unrestored posterior proximal surfaces 'at risk'. These other proximal surfaces may already, even in the absence of clinical and radiographic evidence, display an incipient (white spot) lesion. Clinical trials suggest that multiple applications of varnish to such at risk smooth surfaces may achieve a 75% reduction in caries attack rate.

Fluoride solutions

A range of fluoride-containing solutions have been developed for topical use, including NaF, APF, amine fluoride, iron-aluminium-fluoride, iron-aluminium-manganese-fluoride, stannous fluoride and silver-fluoride. The latter three have been used for arresting proximal enamel and cervical root surface caries, although problems with stability and discolouration have occurred with all three. These issues have been addressed through chemical modifications such as the deletion of manganese and the adjunctive use of potassium iodide after silver fluoride. Stannous and silver fluoride solutions exert anti-bacterial actions. This is a major aspect of their role when used for treating deep dentine caries after excavation and before restoration, however to date they have not been used widely in clinical practice. Clinical studies have shown that single applications of 10% SnF_2 (for 30 seconds) repeated at six monthly intervals over a period of two years can have a dramatic effects in terms of caries arrest on proximal caries in posterior teeth, when used in conjunction with at-home twice daily use of a fluoridated dentifrice (0.4% SnF_2).

Fluoride-releasing restorative materials

Although not designed as a primary preventive measure, fluoride-releasing restorative materials located on tooth surfaces adjacent to those affected by incipient caries contribute fluoride to saliva and plaque fluid and thereby exert beneficial effects by promoting remineralisation. Of the materials commercially available at the time of writing, the highest fluoride release has been documented with the glass ionomer material Fuji VII. Glass ionomer materials can be recharged when concentrated fluoride products are applied topically their surfaces, for example toothpastes and self-applied gels. Fluoride release from compomers and composite resins is very small in comparison, and typically is based on dissolution rather than ionic exchange, meaning that recharging is not possible.

Enhanced fluoride uptake

Several techniques have been developed to specifically enhance the uptake of fluoride into the outer layers of the enamel, which will have benefits in terms of both dental caries and dental erosion. Iontophoresis employs a small electrical current to displace charged ions into pores within enamel. Laser-activated fluoride employs a non-destructive beam of laser energy to destabilise the enamel surface and trap fluoride within the surface by both physical and chemical means. Penetration of fluoride ions into enamel, and their retention over periods of several weeks (as shown by electron probe microanalysis) is increased markedly by laser treatment. This effect was first documented in the early 1970's, but was not subjected to intense investigation until the mid 1980's. Laser irradiation using the appropriate wavelengths can alter the organic and mineral components of the tooth surface, and create a surface that is more likely to uptake fluoride, calcium and phosphate ions. Immediately beneath the surface, micro-spaces and micro-sieves are created, which acts a reservoir of mineral phases, and impair mineral loss. This augments the surface changes that increase the resistance of the tooth surface to acid and cariogenic attack. In the presence of high concentrations of fluoride, the additional possibility exists of conversion of hydroxyapatite to fluorapatite. Together, these chemical and physico-chemical changes bolster the defences of the tooth surface against acid attack.

The combination effect of lasing and fluoride confers upon treated enamel a remarkable increase in acid resistance, when assessed in laboratory models of dental caries and dental erosion. The published literature suggests that slightly better results are obtained for visible lasers when the enamel or root surface site is first treated with fluoride (either neutral sodium fluoride, or acidulated phosphate fluoride) and then lased, compared with the reverse sequence.

Using laser-enhanced fluoride therapy, the dental practitioner can alter the likelihood of caries initiation and caries progression in a patient with high caries risk. Even should caries develop and progress, the depth of the lesion at a lased enamel or root surface site will be reduced by as much as one half compared with an unlased site. Laser preventive therapy can be applied to prevent caries developing, or to reduce the progression rate of existing lesions on the enamel and root surfaces of teeth. Most studies have reported reductions of between 31 and 50% in caries initiation and progression. Clinically, this method has been targeted at high risk patients with impaired salivary parameters, or patients with cervical dentinal hypersensitivity where dissolution of tooth structure occurs because of depressed resting salivary pH.

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Part 2 will follow in the next edition of Synopses.

Gingival overgrowth conditions in children and adolescents

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Introduction

Gingival overgrowth (GO) is a term used to describe the enlargement of gingiva without committing to a histologic diagnosis. Hyperplasia is a histological term used to describe the increase in the size of an organ due to an increased number of cells and should be distinguished from hypertrophy which is an overgrowth due to increased size of the cells.[1] Histometric analysis of drug-induced GO has shown that the GO lesion is characterised typically by an increase in the normal growth (identical numbers of fibroblasts and amount of collagen were found per section of phenytoin-induced GO tissue and normal gingival tissue) as opposed to a cellular hypertrophy or hyperplasia,[2] hence the accepted term gingival overgrowth (GO).[3] GO usually starts in the interdental papilla and spreads to involve the marginal gingiva. In severe cases, the enlarged gingiva may cover the incisal and occlusal surfaces of the teeth.[4]

This narrative review will address the pathogenesis, complications and management of drug-induced GO (DIGO), describe the genetics and clinical presentations of hereditary gingival fibromatosis (HGF) and neurofibromatosis I (von Recklinghausen disease), and discuss some systemic diseases which may present as GO.

Drug Induced Gingival Overgrowth (DIGO)

Today, an increasing number of children are receiving solid organ transplants such as kidney, liver, heart and lung. For a successful transplant, the compatibility of the antigens associated with the blood type (ABO system) and human leucocytes antigen (HLA) is the most important factor.[5] Before transplantation, the blood or tissues of the donor and the recipient must be matched to prevent hyper-acute rejection due to preformed antibodies between the donor and patient. Immediately before and after the transplant, and thereafter as long as the graft is functioning, the recipient also needs immunosuppressive treatment.[5] In the absence of immunosuppression, transplanted organs will undergo progressive immune-mediated injury.[6] This immunosuppression regime usually comprises a triple therapy: a combination of corticosteroids such as prednisolone, calcineurin inhibitors such as cyclosporin or tacrolimus, and lymphocyte-proliferation inhibitors such as azathioprine, which is a non-steroid.[5]

Cyclosporin (CSA) is the most common immunosuppressant used to prevent graft rejection and is aimed mainly at the cell-mediated immune response. Cyclosporin inhibits T-cell function, while having a minimal effect on humoral immunity. However, CSA has side effects such as nephrotoxicity, hepatotoxicity, hypertension, neurotoxicity, tremors, hypertrichosis and hirsutism. Severe GO is also a well-known side-effect associated with CSA. Hypertension, a common finding in CSA-treated patients, is often treated with nefidipine, diltiazem and verapamil, which have also been associated with GO. Investigators have found an increase in the prevalence and/or severity of GO in patients concomitantly medicated with nefidipine, suggesting a synergistic effect.[7-10] The combination of CSA, prednisolone and azathioprine has an antagonistic effect, reducing the severity of DIGO.[11-13] Replacing CSA therapy with tacrolimus has been shown to reduce the severity of GO compared to patients medicated with CSA.[14,15] A study of 27 pediatric subjects receiving tacrolimus found that none experienced GO.[9] This change was however diminished when a calcium channel blocker such as nefidipine was used concomitantly with tacrolimus.[9,15]

The drugs most commonly involved in DIGO are CSA, phenytoin and nefidipine. Sodium valproate has been implicated in isolated case reports and a recent cross sectional study.[16,17,18] Central nervous system stimulant drugs such as amphetamines, used as part of the multimodal management of attention deficit hyperactivity disorder in children, have also been shown to be significantly associated with gingival enlargement.[19]

Prevalence

The prevalence of GO varies in different studies, depending on the drugs used, the methodology used for diagnosing and classifying GO and the population tested. The prevalence of GO is approximately 40-57% in patients taking phenytoin,[20-22] 43-85% in patients taking CSA,[12,23-27] and 6-15% in patients taking calcium channel blockers such as nefidipine,[28,29] verapamil30 and amlopidine.[31] The prevalence of GO in the general population is approximately 2% with a higher incidence in children and male patients.[32] Males were found to be three times more likely to develop GO than females.[29]

Clinical presentation

Clinically, the gingiva appears pink and bulky with loss of stippling. The gingiva appears more fibrous and less inflamed than non-specific inflammatory, hormonal or leukaemic-type gingival enlargements and has a color similar to surrounding mucosa. However, secondary inflammation may induce edema, ulcerations and bleeding on probing. Despite the pharmacodynamic differences between the DIGO-inducing drugs, the lesions produced are clinically similar in appearance.[33]

Pathogenesis

The pathogenesis of DIGO appears multifactorial and the result of interactions between the drug or its

metabolites, and fibroblasts in the gingival tissues. All DIGO-inducing agents appear to lower cytosolic free calcium ions and systemic and local folic acid concentrations, both of which are associated with a reduction in collagenase activity and connective tissue catabolism.[11] The use of systemic folic acid (5mg/day) with phenytoin has been shown to delay the onset and reduce the severity of GO induced by phenytoin.[34]

There are three significant factors important in the expression of these gingival changes. These are drug variables, plaque-induced inflammatory changes in the gingival tissues and genetic factors.[3] Genetic factors determine the heterogeneity of the gingival fibroblast and can also influence drug metabolism, pharmacokinetics and pharmacodynamics.[3] The drugs can influence the inflammatory response, by affecting the nature of the cellular infiltrate or the release of cytokines, prosta-glandins and growth factors.[3] Inflammatory changes within the gingival tissue can in turn orchestrate the interaction between the drug, fibroblast and the subsequent activity of the cell.

Drug-induced alterations in gingival connective tissue homeostasis

The predominant cell type in the gingiva, the gingival fibroblast, is responsible for the synthesis and turnover of collagen and glycosaminoglycans (GAG) in the extracellular matrix.[35,36] All forms of DIGO have a common histological alteration. There is an increase of the connective tissue extracellular matrix, which can be an increased production of extracellular matrix protein or a misbalance in the connective tissue turnover or both. When stimulated by DIGO-inducing drugs, gingival fibroblasts exhibit altered proliferative responses with increased proliferation, increased collagen production and decreased matrix metalloproteinase (MMP) activity at affected sites.[27] Collagen production from gingival fibroblasts is controlled by the synthesis and release of MMPs and tissue inhibitors of metalloproteinases (TIMPs). Matrix metalloproteinases are endopeptidases known for their ability to cleave extracellular matrix molecules. Other studies have shown that phenytoin and nefidipine can alter the gene expression for collagen synthesis, which may be an important factor in the pathogenesis of GO.

An enhanced expression of type IV collagen genes and an increased deposition of type IV collagen has been shown in patients with phenytoin and nefidipine-induced GO.[37] The high expression of Type IV collagen might explain the increased production and reduced turnover of extracellular matrix proteins because Type IV collagen is resistant to bacterial collagenases and tissue MMPs and could remain in the tissue.[37,38]

The connective tissue in phenytoin-induced GO has a significantly higher volume density of non-collagenous matrix than collagenous matrix, suggesting that inducing drugs are also able to directly stimulate gingival fibroblasts to increase GAG synthesis.[35,36,39] *In vitro* studies have also shown that the extracellular matrix produced by fibroblasts stimulated by phenytoin facilitate fibroblast spreading.[40]

The role of growth factors and cytokines which control connective tissue homeostasis has also been reported. The local fibrosis in DIGO has been attributed to the ability of CSA to specifically stimulate T-cell transforming growth factor (TGF)-ß transcription.[41] Enhanced TGF-ß production results in a decrease in MMPs and upregulation of TIMPs, accounting for reduced proteolytic activity in fibroblasts and favoring the accumulation of extracellular matrix.[42] Other investigators have shown that when monocytes and phagocytes are incubated *in vitro* with phenytoin or CSA, expression of platelet derived growth factor is increased. Platelet-derived growth factor is a chemo-attractant for fibroblast proliferation and upregulates the synthesis of GAGs, fibronectin and collagen.[43-45] Phenytoin and CSA also upregulate the production of interleukin-6 and interleukin-8 in human gingival fibroblasts which contribute to the enhanced recruitment and activation of inflammatory cells.[46,47] Interleukin-6 is also able to enhance proliferation of fibroblasts as well as upregulate collagen and GAG synthesis. Immunohistochemistry studies have also shown that keratinocyte growth factor is elevated in CSA-induced GO, suggesting that it may have a role to play in the enhanced epithelial proliferation associated with GO.[48,49] Fibroblasts from phenytoin-induced GO also appear to possess marked deficiencies in phagocytosis. This reduced phagocytic activity has been suggested as a contributing factor to unbalanced degradation and fibrosis.[50,51]

Risk factors influencing the severity of GO

Age

Age has been implicated as an important predisposing factor because DIGO has been observed to be more prevalent in children and adolescents than in adults, and more prevalent in males than females.[52] Gingival fibroblast androgen metabolism was increased when stimulated by nefidipine and CSA, readily metabolising testosterone to 5-ά-dihydrotestosterone. This metabolite had a stimulatory effect on the synthetic activity of fibroblasts and could cause an increase in collagen synthesis and/or a decrease in collagenase activity. This could be significant in the pathogenesis and increased propensity of DIGO in children and adolescents because circulating androgen levels will be higher in adolescents.[52] Age at transplantation and duration of CSA therapy have also been suggested as risk factors for DIGO[10,24,26,53,54] but other authors have disputed this.[27] The greater ability of fibroblasts of younger patients to multiply and generate collagen has been proposed as an explanation.[53]

Somacarrera *et al* (1994) and Kilpatrick *et al* (1997) found that the severity of GO was inversely related to age at the time of transplantation; the most severe GO was found in children who had been transplanted at a younger age and had been on immuno-suppressive therapy for the longest periods.[12,54] However, Hosey *et al* (2002) found that GO decreased with the duration of CSA therapy.[24] This may be because younger children tend to need higher doses of CSA to achieve immunosuppression after grafting because children have lower bioavailability and increased plasma clearance, together with a more limited absorptive surface area in the small intestine.[24] The reduction of GO with increasing duration of CSA therapy may also be due to improvements in oral hygiene, especially as children who had been transplanted the longest are most likely to have had the greatest contact with the dental team.[24]

Serum concentration and dosage

The relationship between the prevalence and extent of GO and drug pharmacokinetic variables is still unclear. A certain threshold concentration of the drug or its metabolite may be necessary to 'activate' gingival fibroblasts. Below this level, the biological effect of the drug or its metabolites on the gingival tissues is dispersed; similarly, increasing the levels of the drug above this threshold will not increase the severity of the lesion.[53] Accordingly, some authors have found no correlation between GO and CSA blood levels.[8,14,24,26,55,56] However, others have contested that dose and serum levels of CSA are significant risk factors for the development and severity of GO.[11-13,21,27,57] Admittedly, many of these studies are small, cross-sectional studies. In a larger prospective longitudinal study, a group of 100 patients was followed in the first six months post-transplant and a positive correlation between CSA blood concentrations and GO was found.[12] In this study, all patients were subject to an oral hygiene training and motivation program and plaque and gingivitis levels decreased significantly throughout the study period.

Oral hygiene and periodontal variables

There is considerable evidence for a positive relationship between inflammation and GO.[3] The inflammatory changes induced by plaque can modulate interactions between the drug and gingival fibroblasts.3 Most cross sectional studies have found significant positive correlations between plaque scores, gingival inflammation and GO.[10,25,27,29,33,57] It is unclear if the relationship between poor oral hygiene, plaque accumulations and GO is causal or incidental because the association between plaque and DIGO may arise through a reduced ability to perform effective oral brushing. When evaluating these studies, it is also important to consider that subsequent inflammation is dependent on the individual's susceptibility to plaque pathogens and may be muted as a result of immunosuppressant therapy.[5,58]

It has been suggested that the plaque and gingivitis-mediated risk for the extent and severity of GO is indirectly related to the dose of CSA because the local bioavailability of drugs is markedly increased by inflammation and plaque.[27,59] Within the gingiva, CSA can induce extracellular matrix deposition by stimulating local T-lymphocytes to release TGF-ß,[41] which can act on fibroblasts to increase collagen synthesis, inhibit MMPs and upregulate TIMPs. The importance of local inflammation in the development of GO is also witnessed by the site specific nature of the GO, with lesions principally affecting the labial surfaces of the anterior teeth.[3,7,27,33,53] This may be because the anterior region is most susceptible to trauma and mucosal dryness.[33]

It has been shown that patients on azathioprine and prednisolone have lower GO scores.[12,13] This could be a manifestation of the anti-inflammatory properties of azathioprine and prednisolone, which would support the role of inflammation as a true risk factor for GO or it could be related to lower CSA concentrations used in the azathioprine group.[11-13] Good plaque control and reduction of gingivitis are important goals in the management of DIGO because they help to minimise the severity of GO by removing the inflammatory component of the lesion.[13,60,61,62]

Concomitant medication

There is evidence that the combination of nefidipine and CSA in organ transplant patients results in more severe GO than if each drug was used singularly.[7-10] Other drugs taken by organ transplant patients may also modify the expression of DIGO. In children, dosing with azathioprine and prednisolone appeared to confer some protection against the development of severe GO which may be due to the anti-inflammatory actions of these drugs on plaque-induced gingival inflammation.[11-13]

Polypharmacy with multiple anticonvulsant drugs may exacerbate phenytoin-induced GO. Phenytoin is hydrolysed by hepatic cytochrome P-450 enzymes to a metabolite (4-HPPH) that is able to induce GO. When other anticonvulsants such as phenobarbitone, primidone and carbamazepine are given together with phenytoin, they stimulate the hepatic P-450 isoenzyme and are all metabolized to 4-HPPH. It is the resultant increase in serum levels of this metabolite which is capable of inducing GO.[63]

Genetic predisposition

Not all patients on phenytoin, CSA or nefidipine therapy develop GO. In clinical studies, patients are referred to as 'responders' or 'non-responders' based on the absence or presence of GO. Such inter-individual susceptibility to these gingival changes may be related to a patient's susceptibility to this unwanted effect.

One of the genetic factors implicated is the response of gingival fibroblasts to phenytoin, CSA and calcium channel blockers. Fibroblasts from different strains have shown heterogeneity in response to CSA or calcium channel blockers. Rates of cell proliferation have been shown to increase, decrease or remain unchanged for different cell strains and for different drug concentrations. Both increases and decreases in protein/collagen synthesis as well as the failure to alter synthesis have been reported.[3] Different fibroblast strains were also found to show marked inter-individual and inter-strain variations in MMP and TIMP activity.[3]

Variations in the enzyme activity of cytochrome P-450 may also affect an individual's response to GO-inducing drugs. Cytochrome P-450 genes exhibit considerable genetic polymorphism which result in inter-individual variation in levels of enzyme activity.[3] Investigation of the human lymphocyte antigen (HLA) expression has also shown that there may be a link between particular HLA antigens and the expression of GO.[7,64] There is evidence that genetic markers such as HLA-B37 and HLA-DR1 confer some protective effect and patients exhibiting these genetic markers experience less severe GO.[7,64]

Complications

DIGO is not only disfiguring but in paediatric recipients, it may interfere with normal oral development and function. DIGO may cause delayed and/or ectopic eruption of teeth, impaired speech, unpleasant appearance which may have a psychological effect on children and difficulty in maintaining optimal oral hygiene.[51] Tooth migration has been reported due to severe DIGO.[1] DIGO may also promote plaque accumulation and complicate professional and self plaque control, especially in children with disabilities who are under

anticonvulsant therapy.[1] These children may become susceptible to caries and periodontal diseases. Furthermore, severe GO may impair mastication, disturb occlusal contacts and place young children at risk of malnutrition.

Treatment

The treatment of established GO may be conservative with meticulous professional and personal oral hygiene, and chlorhexidine oral rinses. This may modify the degree and severity of GO by directly resolving any inflammatory component induced by plaque.[51] Sometimes, modification of the dosage or replacement of drugs may be possible. For example, switching to tacrolimus in suitable cases may successfully minimize the risk of developing GO or resolve or reduce existing CSA-induced GO.[9,14,15] However, tacrolimus has been associated with neuropsychological and behavioral side-effects such as anorexia nervosa-like symptoms with weight loss, depression, school problems, imsomnia, aggressive and anxious behavior.[65] In older transplant patients, prednisolone and azathioprine have been shown to afford patients some protection against GO and may reduce the severity of this side effect.

Surgical intervention with lasers or traditional scalpel techniques may be necessary in young children in whom the GO is severe. The timing of surgical intervention is controversial. There is some evidence that GO reduces over time irrespective of oral hygiene.[24,66] Adults who had undergone heart transplantation were followed for 36 months and a gradual and progressive reduction in the sensitivity of the periodontal tissues to CSA was found, suggesting that delaying surgery for at least 3 years may be helpful in reducing the rate of recurrence.[66] A study on pediatric subjects also found that the severity of GO reduced with duration of CSA therapy.[24] However, Kilpatrick *et al* (1997) found otherwise, with the most severe GO found in those who had been transplanted at a younger age and immunosuppressed for the longest duration.[54] The conflicting results may be due to confounding variables such as oral hygiene standards, effects of mouth breathing, imperfections in restorations, drug dosages, the different pharmacokinetic and pharm-acodynamic variables in adults and children and the study methodology used. Nevertheless, early surgery may be indicated in children to improve oral function, optimise dental development and minimize potential psychological problems associated with an abnormal dentition.[51] Meticulous oral hygiene for preventing the recurrence of GO is important after surgery. In general, recurrence is inevitable and patients and parents must be advised.

An additional possibility for the treatment of GO is the use of antibiotics such as azithromycin (AZM), metro-nidazole (MNZ) and clarithromycin. Their mechanism of action has been attributed to antibacterial action, reduction of local inflammation and possible suppression of protein synthesis in the fibroblasts before severe GO is established.[5] Studies have found that neither AZM nor MNZ were able to produce a complete regression of CSA-induced GO, although they were successful in reducing the amount of GO.[67,68] The outcomes following antimicrobial therapy are most likely related to a reduction in the accompanying inflammatory process rather than to a modification of the fibrogenic process in the gingiva.[68]

Preventive therapy is important in reducing the deleterious effect of these drugs on the gingival tissues and should be implemented early, even before drug administration. It is therefore prudent to work closely with the physicians treating such children and implement early preventive programs to minimise gingival inflammation and place patients on more frequent professional recalls to help prevent the onset or retard the occurrence of severe GO. Dentists should also regularly see patients who have been prescribed drugs known to be associated with GO because of the potential for carcinoma to occur in such overgrowths.[69]

Hereditary Gingival Fibromatosis (HGF)

HGF is a generalised enlargement of the gingiva which may have an inherited pattern, be part of a more extensive syndrome, or be idiopathic and presenting as an isolated condition. The condition is inherited in an autosomal dominant pattern although autosomal recessive forms have been reported. Some cases are sporadic mutations. In the autosomal dominant form, patients present with hypertrichosis, craniofacial deformities, mental disability and epilepsy, but in most patients the only clinical manifestation is gingival fibromatosis. HGF is considered the most common syndromic gingival hyperplasia and may be a feature of syndromes such as Zimmerman-Laband, Rutherford, Cross, Murray-Puretic-Drescher, Schinzel-Giedion, Cowden and Prune-Belly syndromes.[1]

Clinical presentation

HGF is characterised by an excessive accumulation of extracellular matrix resulting in generalised, irregular and fibrotic enlargement of the gingiva.[42] Enlargement is painless and slowly progressive and dependent to a great extent on the oral hygiene of the individual. Clinically, the enlargement is usually pink, firm, non-hemorrhagic and non-exudative but inflammation and edema may make some areas edematous and erythematous. In severe cases, the enlargement may completely cover the teeth and may be found in other oral sites including the mandible, buccal mucosa, tongue and submandibular tissue.[70]

Histological presentation

Histologically, there is a large amount of collagen and fibroblasts. The gingiva is characterised by dense connective tissue rich in collagen fibres, covered by hyperplastic epithelium with extremely long rete pegs and few or no inflammatory infiltrates, unless gingivitis or periodontitis is present. Histology is not specific and the diagnosis is based on the history of the disease and on clinical manifestations.[1]

Pathogenesis

The fibroblast cell proliferation rate in HGF tissue is significantly higher compared to normal gingival cells, translating to greater amounts of collagen and extracellular matrix formed.[71] Fibroblasts from HGF gingiva have reduced MMP levels, suggesting the gene defect in HGF causes an impairment of the balance between MMPs and TIMPs which regulate extracellular matrix turnover.[42] Impaired collagen phagocytosis due to significantly lower proportions of phagocytic cells may be a mechanism contributing to fibrosis.[50]

Complications

The most common complications of HGF are tooth migration, prolonged

retention of primary teeth, delayed eruption and diastema. Sometimes, severe GO covers the crowns of the teeth and results in esthetic and functional problems such as abnormal occlusion, inability to close the lips and impairment of speech and mastication. Due to difficulties in professional and personal plaque control, the individual is at risk of severe caries and periodontal disease.

Management

Treatment involves improvement in the oral hygiene regime with frequent professional and personal oral hygiene practices. Gingivectomy is sometimes required to allow the progressive eruption of the permanent dentition, improve cosmetics and restore function. However, recurrence is common even after complete surgical excision. Some authors have suggested that there is less chance of recurrence if the gingivectomy is delayed till the permanent dentition is in place.[72] However, delaying the gingivectomy with the goal of avoiding recurrence is contraindicated in some cases, such as when deciduous teeth are retained and permanent teeth are entrapped within overgrown gingival tissue.[73]

Neurofibromatosis 1 (NF-1, von Recklinghausen neurofibromatosis)

NF-1 is a hereditary autosomal dominant disease that is the most common single gene disorder to affect the human nervous system; spontaneous mutations have been reported. The estimated incidence is 1:2,500.[74] NF-1 is caused by a mutation of the gene on chromosome 17 that codes for the protein neurofibromin, which is thought to have a role as a tumor suppressor. The disease develops gradually and clinical manifestations increase over time. For example, although most individuals who develop NF-1 are not born with café-au-lait spots, these skin lesions develop during the first three years of life, prompting parents to seek medical advice. The disease is also expressed in exacerbations, usually during growth, puberty and pregnancy. Cutaneous neurofibromas do not usually develop till pre-adolescence but there is increasing tumor load during adolescent and young adult years (puberty).

The most common lesions are multiple skin fibromas, axillary freckling, Lisch nodules, bone malformations and café-au-lait spots, which are the result of proliferation of cutaneous nerve endings. These are considered major disease manifestations of NF-1 and are invaluable in making or excluding the diagnosis. Café-au-lait spots are irregularly shaped but smooth bordered and evenly pigmented brown macules. The smooth border differentiates them from lesions seen in other hyper-pigmentation disorders such as McCune-Albright syndrome. Lisch nodules are hamartomas of the iris and their incidence in NF-1 increases with age.

Neurofibromas can arise from either a single cell or more than one mutated Schwann cell. They are benign tumors composed of Schwann cells, fibroblasts, mast cells and vascular components. Three subtypes of neurofibromas exist; cutaneous, subcutaneous and plexiform. Both cutaneous and subcutaneous lesions are circumscribed and may be brown, pink or skin colored but neither is specific for NF-1. The plexiform neurofibroma, which consists of hypertrophic nerves arranged as lobules in the connective tissue, is pathognomonic of NF-1. While cutaneous neurofibromas may become a major cosmetic problem, they are not premalignant and do not transform into malignant tumors. On the other hand, plexiform neurofibromas are of significant concern because of the potential for cosmetic disfigurement; even though they are benign tumors, their growth can be aggressive and progressive and can undergo malignant transformation.

Neurofibromatosis 2 is a different clinical entity with only intracranial and intraspinal lesions and does not develop any form of gingival hyperplasia.

Complications

Complications of NF-1 include mental handicapping, speech impediment, headaches, seizures, psychological disorders and hypertension. The GO can cause impaction and malposition of teeth, poor oral hygiene and increased incidence of caries and periodontal disease due to difficulties in plaque control. The lifespan of these patients is significantly shortened and causes of death are usually cancer, myocardial infarction, cerebrovascular accidents or pneumonia.

Leukaemic gingival infiltrates

Leukaemia, especially monocytic leukaemia, can involve the gingiva by infiltration with immature white blood cells.[75,76] These patients may develop a generalised GO secondary to massive infiltration of neoplastic white blood cells, presumably because these cells retain a certain amount of chemotactic ability and are drawn to an area of inflammation such as gingivitis. Clinically, the gingival enlargement is generally less fibrotic and appears soft and friable and is more edematous and erythematous. Histologically, atypical and immature white blood cells (blasts) are seen and indicative of malignant infiltration.[70] Leukaemic infiltrates respond when the underlying systemic disease is treated and the local factors including oral hygiene are improved.

Gingival hyperplasia as a manifestation of Hodgkin's lymphoma (HL)

This is a lymphoid malignancy which usually occurs in children and young adults and is characterised by the presence of Reed-Sternberg (RS) cells, derived from B-lymphocytes. Patients usually present with swollen but non-painful lower cervical and/or mediastinal lymph nodes; this may be accompanied by fever and weight loss. Gingival enlargement is a paraneoplastic manifestation and can precede the diagnosis of HL. A recent case report described the first case of GO, premature root resorption and alveolar bone loss, which preceded the diagnosis of HL in a nine year old boy.[77] Clinically, the gingiva appears inflamed and hyperplastic. Histologically, the gingival biopsy will disclose an inflammatory infiltrate characterised by RS cells, which have a bilobed nucleus (resembling an owl's eye) with prominent eosinophilic nucleoli. Hodgkin's lymphoma responds well to chemotherapy and regression of the GO is usually seen with remission of the lymphoma.

Conclusion

Gingival overgrowth is a condition that may be seen in the paediatric population, particularly in subgroups of the paediatric population who depend on systemic medications such as phenytoin, CSA and nefidipine. There is also increasing evidence that sodium valproate and amphetamines

are implicated. DIGO is more frequent in children and adolescents and genetic and environmental factors such as age, drug dosages, therapeutic regimes, duration of drug therapy and oral hygiene are predisposing factors that can modify the final outcome of GO. Stringent oral hygiene measures may reduce the severity of overgrowth, but may not inhibit its development. Hence, the management of GO is a challenge to the clinicians involved in the care of these patients.

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Appendix I: Syndromes associated with generalised gingival fibromatosis

Byars-Jurkiewicz Syndrome	GF, hypertrichosis, kyphosis, giant fibroadenomas of breast
Cross Syndrome	GF, microphthalmia, mental retardation, hypopigmentation
Laband Syndrome (Zimmerman-Laband Syndrome)	Rare autosomal dominant disorder GF, ear, nose, bone, nail defects, hepatosplenomegaly (see Holzhausen M, Goncalves D, Correa FOB, Spolidorio LC, Rodrigues VC, Orrica SRP.A case of Zimmerman-Laband Syndrome with supernumerary teeth J Periodontol 74(8):1225-1230,2003)
Murray Syndrome	GF with juvenile hyaline fibromatosis[77]
Ramon Syndrome	GF, hypertrichosis, cherubism, mental and somatic retardation, epilepsy
Rutherford Syndrome	GF, corneal dystrophy
Prune-Belly Syndrome	GF, hypoplastic abdominal muscle, obstructive nephropathy[83]
Jones-Hartfield Syndrome	GF, sensorineural hearing loss
Schinzel-Giedion Syndrome	Autosomal recessive disorder[85] GF, craniofacial dysmorphism, club feet, cardiac and renal malformations
Proteus Syndrome	Sporadic mutation with multifocal overgrowth[84]
Cowden Syndrome	Gingival, oral and facial papillomatosis
Murray-Puretic Drescher Syndrome	GF, hyaline fibromas of head, trunk and extremities
Lysosomal storage disease eg Hunter, Hurler, Morguio, Maroteaux-Lamy and Sly Syndromes, I cell disease	Specific enzymatic deficiencies Childhood GF, wide alveolar ridges, widely spaced teeth
GF with growth hormone deficiency	
GF, hypertrichosis, epilepsy and mental retardation syndrome	GF, universal distribution of terminal hypertrichosis, coarse facies (in some variants, intelligence can be normal)[71,72]

Australian and New Zealand Society of Paediatric Dentistry

SA Branch

An invitation is extended to all dental personnel interested in the promotion of children's dental health to join us at two half-day clinical seminars in 2006.

Seminar 1: Cleft Lip and Palate Conditions

12.30pm – 5.00pm Monday, 28 August
at Next Generation, Memorial Drive, Adelaide

RSVP Monday, 7 August

Guest speakers

- Associate Professor Nicky Kilpatrick, Director, Department of Dentistry, Royal Children's Hospital, Melbourne
- Mr Mark Moore, Head, Department of Plastic Surgery, Women's and Children's Hospital
- Ms Ros Selles, Speech Pathologist, Australian Craniofacial Unit, Women's and Children's Hospital

Seminar 2: The Medically Compromised Paediatric Patient and Dental Management

8.30am – 1.00pm Friday, 24 November
at Next Generation, Memorial Drive, Adelaide

RSVP Friday 3 November

Guest speakers

- Dr Chris Pearson, Head, Department of Paediatric Medicine, Women's and Children's Hospital
- Dr Christina Boros, Head, Department of Paediatric Rheumatology, Women's and Children's Hospital
- Dr Heather Tapp, Staff Specialist, Department of Oncology and Haematology, Women's and Children's Hospital
- Dr Sam Gue, Head, Department of Paediatric Dentistry, Women's and Children's Hospital

Cost for each seminar

ANZSPD member	$130
Non-ANZSPD member	$180
Therapists & hygienists	$140
Students	$140

For further information and registration forms please email ANZSPD (SA Branch) secretary Dr Mary Apps: apps_mvb@yahoo.com.au

Pre-conference course

Wednesday, 6 September (9am-4pm)

(L) Dr John Winters

(R) Dr Kathy Harley

The Biennial NZDA conference ('The Business of Smiles') is being held in Auckland from 6-9 September. The NZ branch of ANZSPD is privileged to have Dr Kathy Harley and Dr John Winters visit Auckland and present at a pre-conference course on Wednesday, 6 September. The theme for the day is 'Managing Dental Anomalies – a lifetime perspective'. These lectures are NZDA accredited.

Kathy Harley graduated from Guy's Hospital Dental School, University of London in 1981. She gained her Masters degree in Conservative Dentistry in 1986 and was appointed Consultant and Honorary Senior Lecturer in Paediatric Dentistry at the Eastman Dental Hospital in 1991 where she concurrently held the appointment of Honorary Senior Lecturer at the Eastman Dental Institute. She took up the post of Consultant in Paediatric Dentistry at the Edinburgh Dental Institute in September 1999 where she is Head of Department. Her topics will include:

- Hypodontia (including loss due to trauma) – restoration of the missing unit, partial dentures for the very young, teenage alternatives, treatment planning issues
- Amelogenesis/Dentinogenesis Imperfecta: from childhood to adulthood

John Winters graduated with a Masters of Dental Science from the University of Western Australia in 1989. He is a Consultant Paediatric Dentist and Chairman of the Dental Department, Princess Margaret Hospital for Children in Perth. He also runs a successful private practice utilising modern technology. He has written a number of articles including a chapter on Paediatric Dental Care in the Mckinstry textbook on Cleft Lip and Palate. John is currently the Federal President of the ANZSPD. His presentations will include:

- Practical complete oral rehabilitation under GA
- Digital intra-oral photography in dentistry
- The role of general dental care in the management of cleft lip and palate. This lecture covers neonatal assessment, natal teeth, pre-surgical orthopaedics, early intervention and preventive care, restorative considerations, and preparation for secondary alveolar bone graft.

Registration fees: $250 – ANZSPD members; $350 non-ANZSPD. All attendees need to have registered with the NZDA conference, Contact: Michaela@conference.co.nz

ANZSPD Federal Secretary-Manager's report

Dr Joe Verco has advised the National Foundation of Ectodermal Dysplasia has an active and comprehensive website which is well worth a visit. The Foundation has been in existence for 25 years and has a myriad of different activities. It can be accessed: http://www.nfed.org

'ANZI' As I had reported in the previous Synopses, the Federal Council decided to apply for registration of the ANZI Character trade mark. I am pleased to report that this process is reaching a satisfactory conclusion. In April we were advised that the Australian Trade Marks Office has deliberated and that our application will be able to proceed to acceptance for the following services:

Class 41

Education and entertainment services relating to dentistry; the organisation of conferences and events.

Class 44: Dental services

The Patent and Trade Mark Attorneys we had consulted, Spruson and Ferguson, have advised this coverage should give sufficient protection for the use of the ANZI Character trade mark.

The Federal Council of A.N.Z.S.P.D. Inc. will be meeting in Dunedin, New Zealand on Thursday, 6 July 2006. This meeting is being held at that time so as to coincide with the annual Australasian Academy of Paediatric Dentistry meeting.

As will be reported elsewhere in this edition, the 2006 R.K. Hall International Visiting Lecturer Tour has occurred. Katie Ayers has been honoured this year as the Lecturer, with visits to Brisbane, Hobart and Adelaide in early March. In the planning and organising of the Tour, it appeared the Gods were determined that the Tour would not proceed! An unbelievable set of obstacles continued to appear in our path, the sum total of which meant the Tour brochure was late in being distributed. This meant attendances were less than would have been desired. However, I am advised those who attended were well rewarded with quality lectures from Katie and her supporting speakers. The next R.K. Hall Tour will occur in 2008, with visits to New Zealand, Victoria, New South Wales and Western Australia probably.

Alistair Devlin

ANZSPD – Branch news 2006

Queensland

The branch has had another active year with regular informative meetings and lectures. Our AGM was held in February 2006, at which Dr YH Thong gave us a very informative, relevant and useful lecture centred on various aspects of allergy as applied to paediatric dental practice. Office bearers were elected for 2006, with Dr Robin Smith as President, Dr Steven Kazoullis as Secretary/Treasurer, Dr John Rutar as Federal Representative and Dr Michael Kenwood as Committee Member.

The RK Hall Lecture series was presented at the Hotel Grand Chancellor on 1 March 2006, at which a broad range of topics were covered. Dr Katie Ayres presented topics including 'Water Fluoridation and Fluorides', 'Update in Local Anaesthetics for Children' and 'General Tips and Tricks in Paediatric Dentistry'. Assoc Prof Richard Widmer gave us some insight into Child Management and Relationship Management. Dr Steven Kazoullis outlined some aspects of Dental Erosion in Children.

Our May meeting saw a change in venue for our regular meetings, which will now be held at the new ADA Christensen House, in Bowen Hills. During our first meeting at this venue, Prof Laurie Walsh presented a lecture on 'New Technologies in Children's Dentistry', during which he outlined future trends in the application of technology to Paediatric Dentistry. Aspects of these new technologies included new methods of plaque testing and pH testing and the use of oxygen based destruction (ozone).

Future events for the branch include a lecture by Prof Newell Johnson in August and our upcoming Clinic Weekend.

Steven Kazoullis

Western Australia

Mainly because of the I.A.P.D. Congress in Sydney in November 2005, and the usual busy schedule of members at the end of each year, the 2005 Annual General Meeting of the Branch was not held until February of this year. The following officers were elected:

President: Tim Johnston

Secretary-Treasurer: Alistair Devlin

Federal Councillor: John Winters

Committee Members: John Camacho, Kate Dyson, Mark Foster, Peter Gregory, Theo Gotjamanos, Peter Readman, Trudy Stewart, Vanessa William.

A motion of congratulations to Professor Theo Gotjamanos was carried by acclamation. Theo has recently been appointed to the Chair of Pathology at the University of Notre Dame in Fremantle.

The formal meeting was then followed by a retrospective on the scientific programme from the I.A.P.D. Congress in Sydney. This post-mortem was dominated by a discussion on the choice of medicament for use in pulpotomies in primary teeth. John Winters started the ball rolling with a compelling presentation in favour of the continued use of formocresol, whilst Vanessa William and John Camacho spoke on what is happening in Victoria and New South Wales respectively. Tim Johnston presented the case for changing from formocresol. All in all, it was a riveting and sometimes passionate session which has given the activities of the branch a kick start for 2006.

The second meeting of the year gave members of the branch the opportunity to hear from Vanessa William and John Camacho. Vanessa and John have both completed their Masters programmes in recent times, Vanessa in Melbourne and John in Sydney. Both presented on their Masters research projects. Vanessa spoke on 'Bonding to Hypomineralised Enamel. Does it work?' with the conclusion that it does but not very well. John spoke on 'Novel DNA-BrdU Labelling Patterns'. John prefaced his presentation by stating that he had lightened up the subject matter. It soon became apparent that this was fortunate. Certainly, it clearly demonstrated that the biochemistry/molecular biology degree John had completed before he studied dentistry had made him eminently qualified to pursue this particular line of research. John's presentation also demonstrated how

the knowledge of cell biology has and is continuing to advance.

The next meeting for the Branch will be the Annual Mid-Winter meeting. This will be on Saturday, 29 July at the Bunker Bay Resort, in the south west region of the state. This meeting will be held in conjunction with the South West Convocation of Dentists. The programme will feature Professor Mark Tennant presenting, and this will be followed by the usual 'Pot Pourri' of case presentations by members, which has proved so successful over the years.

In the meantime, the Local Organising Committee for the 15th A.N.Z.S.P.D. Convention has been busily preparing for the meeting in Broome. The Convention will be opening on the evening of Tuesday, 22 May 2007 and a programme has been devised of two half days and one full day over the following three days so that the attendees can also fully enjoy many of the tourist activities this town has to offer. The Convention will wind up with the Dinner on Friday, 25 May 2007. It is planned to have the Convention brochure ready for distribution by July 2006. As a matter of interest, it has been noted that temperatures in Broome in late May 2006 have been consistently around 31 to 32 deg C maximums and 14 to 15 deg C minimums.

Alistair Devlin

Victoria

The Victorian Branch has had two dinner meetings this year. The first in February was presented by Dr Doug Lee, Orthodontist. Doug's topic was 'Tell Me When – Tell Me Why. The Timing of Orthodontic Treatment'. He gave an entertaining and informative presentation exploring some of the relevant issues involved in the planning of orthodontic treatment he has faced at his practice in the Eastern Suburbs of Melbourne. Doug's lecture was preceded by a postgraduate presentation from Dr Theresia Sudjalim, who is enrolled in the orthodontic program at The University of Melbourne. She discussed her study on the use of Tooth Mousse and fluoride in the prevention of white spot lesions during orthodontic treatment. Dr David Manton is overseeing her study.

The second diner meeting was the Des Crack Memorial Lecture and Dr Peter Wong made the long trip to Melbourne via Sydney to give us a lecture 'Sedation and Resuscitation. Can we live without it?' He discussed the complex issues we face as clinicians treating children and discussed his experience as a Paediatric Specialist who routinely uses sedation. Children are not just little adults and we have to be aware how they can react to various techniques and medications. From sedation medications to local anaesthetic the take home message was know your patient's weight and give the appropriate dosages.

Dr Susan Barry presented a review looking at the evidence based assessment for the use of antibiotic use following avulsion. Mostly we prescribe empirically (according to the trauma guru Andreasen). However there is no evidence for this protocol.

At this meeting Dr David Manton had the pleasure of presenting The Des Crack Memorial Prize to the Final year student of 2005 who achieved the highest marks in Special Needs Dentistry. This year it was awarded to Dr Adam Wallace. We also had the presentation of the ANZSPD Victorian Branch Bursary to Dr Lochana Ramalingam for her submission requesting a Dental Wand for the Department of Dentistry, Royal Children's Hospital. Dr Ramalingham shall be preparing a report for Synopses next year on the use of the Dental Wand in her department.

Felicity Wardlaw

Victoria

Picture 1
Dr David Manton presenting the Des Crack Memorial Prize to Dr Adam Wallace

Picture 2
Dr Lochana Ramalingam receiving the ANZSPD Victorian Branch Bursary

Picture 3
Dr Susan Barry, postgraduate presenter, with Dr David Manton

Picture 4
Dr Peter Wong, the Des Crack Memorial Lecturer, and Dr David Manton

Tasmania

The recent R.K. Hall Visiting Lecturer Series visited the isle of Tasmania in early March. On tour were the highly inspirational Dr. Katie Ayers, Richard Widmer and John Winters, the topic 'Finding the Balance – Paediatrics in General Practice'. The venue was located on Hobart's scenic waterfront and would have provided spectacular views of the bay, if only the weather hadn't played it usual games, going from 30 degrees the day before to a nippy 12 and drizzle. The weather however did not deter an outstanding crowd of attendees, and the program covered the dental team's spectrum, ranging from Orthodontists to Dental assistants.

Katie presented a well balanced series of topics on Anaesthetics and Fluoride – which was obviously a controversial topic even down in Tasmania. Richard and John were their usual affable selves, with John keen to stir the pot over Formocresol, and Richard tackling the difficult role of Child management crossing the line into relationship management. The day proceeded at pace with a healthy floor led discussion between speakers, and left everyone with a feeling that there were some very thought provoking issues to be chewed over later.

Later that night, the executive committee felt the need to follow up a few queries with the Guest speakers, and the debate raged into the night, gently assisted by the capable delights of the Hobart dining scene. I hope that the interstate delegates felt that they were able to take a little bit of Tasmania away with their intellectual food for thought, so helpfully provided by the thoroughly entertaining Katie, Richard and John.

Tasha Dodd

New Zealand

One specific goal for 2006 for the NZ branch has been to increase the profile of paediatric dentistry in NZ amongst our colleagues, auxiliaries, therapists and the general community. We are also committed to raising membership as this allows more people being involved in working towards our goal. Our branch has been busy organising and hosting upcoming events: Dr Tim Johnston to visit New Zealand in June and we are sponsoring Dr Kathy Harley to the Biennial NZDA Conference in Auckland, 6-9 September.

During 2005 in Auckland, study groups were held every 3-4 months at night with members from Auckland and some from out of town. These meetings provided peer contact and a forum to share information, case presentations and collaborate treatment planning for difficult cases. This was followed by an informal dinner at a local restaurant (plenty of wine and fun!). These meetings are NZDA accredited. We will resume the study groups later in the year.

Upcoming events: We are fortunate to have Dr Tim Johnston visit New Zealand, and talk at various cities including: Auckland, Dunedin, Christchurch, Palmeston North, Taranaki and Nelson. His talks are aimed at both dentists and dental therapists, and will involve topics such as 'Pitfalls in Paediatric Dentistry; Restorative techniques; New technology – lasers and microscopes; Utilising dental auxiliaries in private practice'. Talks will be supported by local speakers in Auckland (Dr Nina Vasan) and Christchurch (Dr Heather Anderson).

The Biennial NZDA conference ('The Business of Smiles') is being held in Auckland from 6-9 September. The NZ branch of ANZSPD is privileged to have Dr Kathy Harley and Dr John Winters visit Auckland and present at a pre-conference course on Wednesday, 6 September. The theme for the day is 'Managing Dental Anomalies – a lifetime perspective'. These lectures are NZDA accredited.

As we all remember from the IAPD conference in Sydney last year, Dr Kathy Harley is an excellent speaker with great clinical skills to share. Her topics will include:

- Hypodontia (including loss due to trauma) – restoration of the missing unit, partial dentures for the very young, teenage alternatives, treatment planning issues
- Amelogenesis/Dentinogenesis Imperfecta: from childhood to adulthood

We were also grateful to have our Federal President, Dr John Winters attend. His presentations will include:

- Practical complete oral rehabilitation under GA
- Digital intra-oral photography in dentistry
- The role of general dental care in the management of cleft lip and palate. This lecture covers neonatal assessment, natal teeth, pre-surgical orthopaedics, early intervention and preventive care, restorative considerations, and preparation for secondary alveolar bone graft.

This promises to be a very interesting day and a unique opportunity to spend time with two excellent clinicians. Those who are interested should register with the conference organisers as numbers are limited for this day.

Registration fees: $250 – ANZSPD members; $350 non-ANZSPD

Other news: Dr Katie Ayers had a successful tour in Australia as the visiting RK Hall lecturer earlier this year. We heard that her talks were very interesting and informative.

Congratulations to Dr Nina Vasan who had a baby girl named Sasha on 12 April 2006 – both mum and baby are doing well, despite some lack of sleep!

I trust you have all had a great first half of the year and look forward to seeing you at the upcoming events.

Mary Anne Costelloe

Cambodian dental project

Callum Durward, Brenda Ryan and Andrea Doiron

Cambodia is located in Southeast Asia, sharing its borders with Thailand, Vietnam, and Laos. Its population is about 13 million, with 80% rural and 36% living below the poverty line. The life expectancy for males and females is only 53 years. Between 1970 and 1990 Cambodia experienced war, political turmoil, economic collapse, genocide, radical communism, and poverty. During the Khmer Rouge period from 1975 to 1979, over 2 million people died, and the country was reduced to a primitive Maoist agrarian society. All formal education and health services were stopped. In recent years the situation has improved, but Cambodia is still one of the poorest countries in the region. Oral health is one area that has been largely neglected, even though many Cambodians suffer from dental problems, including oral cancer.

There is only one dental school in Cambodia which is part of the University of Health Sciences. In addition there is a school for dental nurses in Kampong Cham province. The Faculty of Dentistry was founded in 1953 along with the Faculties of Medicine and Pharmacy. The first dental training programme was for auxiliary dentists and followed the French system. In 1972, the first course to train dentists began; however, these dental students were unable to complete their course of study due to the political upheaval caused by the Khmer Rouge. Many of the dental students and auxiliary dentists were killed or died of hardship during that period – only 34 survived. They were later upgraded by visiting Vietnamese professors to dentists.

Oral health status

Caries among children in Cambodia is rampant. In Phnom Penh the average 5 year old has over 10 decayed teeth. Very few children receive dental care, and what treatment is provided is usually just extraction. Between 1990 and 2000 a school preventive programme supported by two international NGOs operated in many schools. It comprised weekly fluoride mouth rinsing and daily brushing, but appears to have had minimal impact on the caries rate. The Ministry of Health is hoping to reinstate a school preventive programme again, once a new sponsor can be confirmed. Unfortunately Cambodia still has no water or salt fluoridation, which would clearly be the most effective public health measures. However these are being considered, and are recommended in the National Oral Health Plan

Dental workforce

Cambodia has about 350 dentists, 1000 traditional dentists, and 270 dental nurses. Cambodian dental nurses are rural medical nurses who have an additional years training in basic dentistry (eg ART, LA, scaling and extractions) and oral health promotion. They are trained at a Regional Nursing School, and a large part of their course is spent providing treatment and oral health education in the surrounding rural villages. Following graduation most return to work as bi-functional nurses in remote rural areas where there are no dentists.

Paediatric dentistry teaching at the Faculty of Dentistry in Phnom Penh

The Faculty of Dentistry has a small staff of dedicated Cambodian teachers who work on a salary of less than $US100 a month. Significant progress has been made under the leadership of the present Dean (Prof Suon Phany) in upgrading the school, but it still has a long way to go to reach the standard of most other schools in Asia. Two years ago the paediatric dental clinic was renovated by an American NGO and is now the busiest and best equipped clinic in the school. It is staffed by three overworked Cambodian dentists, lead by the department head, Dr Nhoung Poum Sen.

Several years ago a group of volunteer NZ and Australian paediatric dentists helped conduct Cambodia's first postgraduate dental course (in paediatric dentistry) for 4 Cambodian dentists. This course lead the way for other diploma courses to be offered in periodontics, dental public health and orthodontics – all taught by visiting teams of overseas volunteer dentists. An oral surgery programme is about to begin shortly. Such courses are needed because only one staff member at the Faculty has a postgraduate overseas qualification – a diploma of oral surgery from NZ – and it is almost impossible for Cambodian dentists to get scholarships to study abroad. Building the capacity of the teaching staff at the dental school is a top priority.

Late last year the Dean of the Faculty asked for help to organise a second diploma course in paediatric dentistry, and to improve the undergraduate programme. Interest was expressed by some of the Australian and NZ paediatric dentists in being involved in teaching this new course, and so in February an agreement was signed with the Rector of the University, and 5 Cambodian dentists were selected. They are:

a. Veng Sonita – Works at “Children of Promise” orphanage.
b. Sok Kunthy – He works full-time in the childen's clinic at the Faculty. Originally trained as dental nurse in Vietnam, he recently upgraded to dentist at the Faculty in Phnom Penh. Kunthy has good clinical skills.
c. Keo Sophany – She works in the dental clinic at the National Paediatric Hospital.
d. Soeun Sopharith – A recent graduate who works at the hospital in Battambang, Cambodia's second largest city.
e. Meas Sokha – Also a recent graduate. Works in a private dental clinic (‘Apsara’) which treats many children.

Three of the five are men, and all but one have good English. During 2006 and 2007 a group of volunteer paediatric dentists from Australia and NZ will visit Cambodia every two or three months to teach the postgraduate diploma programme.

The Dean of the Faculty has also invited ANZSPD members (dentists and therapists) to visit Cambodia as volunteers for one or two weeks. They can work in the paediatric clinic, or perhaps in one of the other clinics at the dental school. Most of the students and staff speak English, and greatly appreciate help from overseas visitors. Donations of dental materials, books and journals are also welcome.

There are many good hotels as well as inexpensive guest houses to stay in Phnom Penh. The food is wonderful, and there is great shopping and sightseeing in Phnom Penh and Siem Reap (Angkor Wat). There are flights to Cambodia via Kuala Lumpur, Bangkok and Singapore, with Malaysia Airlines, Thai International, and Silk Air (the regional carrier for Singapore Airlines).

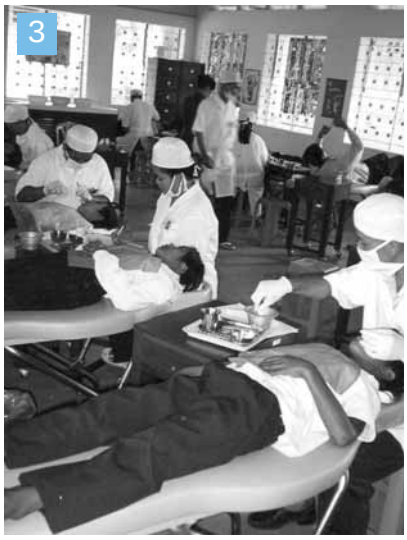
More information about Cambodia and the Faculty can be found on the website: www.oralhealthcambodia.com

There is also a 'Guide for visiting dentists' which has additional useful information and can be emailed to you. You will be met on arrival at the airport by a Cambodian dentist or student, and taken to your hotel. Transport to the dental school can also be organised.

For many people, doing volunteer work like this can be a rewarding and even life-changing experience

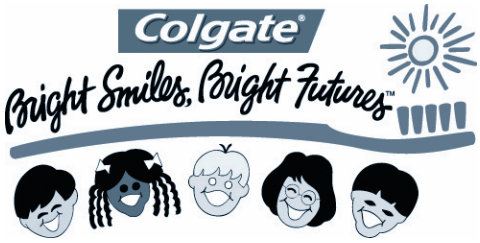
If you are a dentist or dental therapist and interested in volunteering your skills in Cambodia please contact me at: c.durward@clear.net.nz

Pictured below left to right from top left: 1. Dr Poumsen, Head of Paediatric Dentistry (working in old clinic); 2. Cambodian child in old clinic; 3. Clinic at School for Dental Nurses in Kampong Cham; 4. Faculty of Dentistry, Phnom Penh; 5. Rector of University of Health Sciences – Professor Oum Sophal, Dean of Faculty of Dentistry – Professor Suon Phany, Head of Oral Surgery – Dr Hong Someth, Callum Durward; 6. Renovated clinic; 7. First group of graduating paediatric dentistry students with Dean; 8. New group of dentists undertaking the Diploma with Dr Poum Sen



Colgate® Corner

by Barbara Shearer
Colgate Professional
Relations Manager



Dr Rabbit and the Legend of Tooth Kingdom

At the beginning of the year we sent a direct mail to all Year 3 teachers (Year 4 in Queensland) in Australia and New Zealand inviting them to participate in our BSBF Program 'Dr Rabbit and the Legend of Tooth Kingdom'.

We have had a fantastic response to date with approximately 5,500 Year 3 kits, with resources for approximately 176,000 children, being distributed to Primary schools all over the Australia and New Zealand.

These kits are also available to the dental profession by calling our toll free number 1800 075 685 (Australia) or 0800 556615 (New Zealand).

Colgate Oral Health Month

Colgate will again host Oral Health Month in August this year. The purpose of Oral Health Month is to raise oral health awareness in the community, in partnership with dental professionals. Oral Health Month is supported by the Australian Dental Association in Australia and the New Zealand Dental Association in New Zealand. Oral Health Month will feature press releases about oral health issues. Colgate will encourage preschool teachers to conduct oral health education sessions during August. A mail-out to all preschools in Australia and New Zealand will make teachers aware of the Colgate Bright Smiles Bright Futures (BSBF) preschool kits. Encourage preschool teachers in your area to conduct an oral health education session during Oral Health Month. Teachers can request kits by phoning a toll free number 1800 075 685 (Australia) or 0800 556615 (New Zealand) or by ordering online www.colgateprofessional.com.au or www.colgateprofessional.co.nz. Limited stocks of these pre-school kits are also available for use by the dental profession. Oral Health month will also feature in-store promotions and in-surgery promotions.

New Products

We are pleased to announce the arrival of Colgate's new Neutrafluor and Fluorocare Gels. Both contain 5000ppm fluoride and are ideal for patients at high risk of dental decay. Also, the Colgate 360 toothbrush is now available with a compact-sized head. Please see the latest issue of Polish Up for more details.

Changing faces in the Colgate team

After 6 years, Jackie Robinson has left to take up a new role with Australian Health Management. Jackie has been a wonderful ambassador for Colgate. She is extremely well respected in the dental community and her compassionate style of leadership will be missed.

Barbara Shearer has taken up the role of Colgate's Professional Relations Manager for Australia. She is a graduate of the University of Otago (BDS 1986, PhD 1992, MDS 1995) and has worked in academia, private practice and in industry both in New Zealand and the United Kingdom. For the last two years she has been the Professional Relations Manager for Colgate in New Zealand.

We are delighted to announce the appointment of Dr Rebecca Schipper to the role of Professional Relations Manager, for Colgate Oral Care New Zealand.

Dr Schipper will be well known to many members of the Dental Profession in New Zealand. Rebecca has a DMD from Oregon Health Sciences University as well as a BA in Biology. She worked as a general dentist in the US before immigrating to New Zealand in 2003. Most recently Rebecca has held the role of Senior Lecturer and Co-leader of Oral Health Research at the Auckland University of Technology.

Rebecca will be based in the Colgate Offices in Auckland and will commence her role with us on the 26 July 2006.

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Colgate Territory Managers are here to assist you with the products you need in your surgeries.

Contact details for Barbara Shearer, Professional Relations Manager:
barbara_shearer@colpal.com
Office: (02) 9229 5798

Coming events

28 August 2006
Cleft lip and palate conditions

ANZSPD SA Branch
Next Generation, Memorial Drive
Adelaide, South Australia

6 September 2006
Managing dental anomalies – a lifetime perspective

ANZSPD NZ Branch
Auckland, New Zealand

24 November 2006
The medically compromised paediatric patient and dental management

ANZSPD SA Branch
Next Generation, Memorial Drive
Adelaide, South Australia

25 November 2006
Happy Smiles, Happy Children: The Prevention and Management of Common Dental Diseases

ANZSPD VIC Branch
Suma Park Homestead and Conference Centre
Bellarine Highway
Queenscliff, Victoria

23-27 May 2007
ANZSPD Federal Convention

Cable Beach Club Resort
Broome, Western Australia

24-28 May 2007
60th AAPD Annual Session

Henry B. Gonzalez Convention Center
San Antonio, Texas, USA

14-17 June 2007
21st IAPD International Congress

Hong Kong Convention and Exhibition Centre
http://www.iapd2007.com/

8-13 June 2009
22nd IAPD International Congress

International Congress Centre
Munich, Germany

Austalian and New Zealand Society of Paediatric Dentistry

www.anzspd.org.au

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Submissions

All text for inclusion in Synopses must be submitted to the editor on floppy disk, zip disk, CD, or by email. Both PC and Mac formats are accepted. Media will not be returned. Address email to karenkan@optusnet.com.au. Please enclose your contact details and email address with all submissions.

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